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NEWS 1		Web Page URLs for STN Seminar Schedule - N. America
NEWS 2		"Ask CAS" for self-help around the clock
NEWS 3	SEP 09	CA/CAplus records now contain indexing from 1907 to the present
NEWS 4	DEC 08	INPADOC: Legal Status data reloaded
NEWS 5	SEP 29	DISSABS now available on STN
NEWS 6	OCT 10	PCTFULL: Two new display fields added
NEWS 7	OCT 21	BIOSIS file reloaded and enhanced
NEWS 8	OCT 28	BIOSIS file segment of TOXCENTER reloaded and enhanced
NEWS 9	NOV 24	MSDS-CCOHS file reloaded
NEWS 10	DEC 08	CABA reloaded with left truncation
NEWS 11	DEC 08	IMS file names changed
NEWS 12	DEC 09	Experimental property data collected by CAS now available in REGISTRY
NEWS 13	DEC 09	STN Entry Date available for display in REGISTRY and CA/CAplus
NEWS 14	DEC 17	DGENE: Two new display fields added
NEWS 15	DEC 18	BIOTECHNO no longer updated
NEWS 16	DEC 19	CROPU no longer updated; subscriber discount no longer available
NEWS 17	DEC 22	Additional INPI reactions and pre-1907 documents added to CAS databases
NEWS 18	DEC 22	IFIPAT/IFIUDB/IFICDB reloaded with new data and search fields
NEWS 19	DEC 22	ABI-INFORM now available on STN
NEWS 20	JAN 27	Source of Registration (SR) information in REGISTRY updated and searchable
NEWS 21	JAN 27	A new search aid, the Company Name Thesaurus, available in CA/CAplus
NEWS 22	FEB 05	German (DE) application and patent publication number format changes

NEWS EXPRESS	DECEMBER 28 CURRENT WINDOWS VERSION IS V7.00, CURRENT MACINTOSH VERSION IS V6.0b(ENG) AND V6.0Jb(JP), AND CURRENT DISCOVER FILE IS DATED 23 SEPTEMBER 2003
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FILE 'HOME' ENTERED AT 09:34:00 ON 17 FEB 2004

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=> s IL-10 (p) (antibod?)
L1         5599 IL-10 (P) (ANTIBOD?)

=> s l1 (p) (IL-2)
L2         2256 L1 (P) (IL-2)

=> s l1 (p) (Il-2 antibod?)
L3           15 L1 (P) (IL-2 ANTIBOD?)

=> d l3 1-15 bib ab

L3   ANSWER 1 OF 15     MEDLINE on STN
AN   2000114114     MEDLINE
DN   20114114   PubMed ID: 10649854
TI   [Collagen in the treatment of rheumatic diseases--oral tolerance].
     Kolagen v liecbe reumatickych chorob--oralna tolerancia.
AU   Stancikova M; Stancik R; Gubzova Z; Rovensky J
CS   Research Institute of Rheumatic Diseases, Piestany, Slovakia..
     stancikova@vurch.sk
SO   BRATISLAVSKE LEKARSKE LISTY, (1999) 100 (10) 567-71.
     Journal code: 0065324. ISSN: 0006-9248.
CY   Slovakia
DT   Journal; Article; (JOURNAL ARTICLE)
LA   Slovak
FS   Priority Journals
EM   200002
ED   Entered STN: 20000218
     Last Updated on STN: 20000218
     Entered Medline: 20000210
AB   The term "oral tolerance" means antigen specific suppression of immune
     response after oral application of antigen.  Primary mechanisms by which
     oral tolerance is mediated include: deletion, anergy and active cellular
     suppression.  The determining factor in this process is the dose of
     applied antigen.  High doses of antigen develop deletion and anergy of
     cells while low doses of antigen result in bystander suppression.
     Recently bystander suppression has attracted attention in the treatment of
     autoimmune diseases.  This process is connected with induction of
     regulatory T cells of Th2/Th3 phenotypes in gut with characteristic
     profile of anti-inflammatory cytokines as IL-4, IL-10
     and TGF-beta.  By means of circulation the lymphocytes enter the affected
     place and when meeting again with the antigen, they produce the same
     profile of cytokines which they originally made in the gut.  These
     cytokines then suppress local autoimmune and inflammatory reaction
     independently of the antigen type.  After successful trials of treatment
     with low doses of orally applied collagen type II in animal models of
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experimental arthritis, this treatment was also studied in clinical trials in humans with rheumatoid arthritis. Although the results obtained to this date are very promising they can not be considered final. Several questions still need to be solved: identification of responders, determination of character and amount of collagen applied as well as the route of application. Another promising therapeutic approach could be the simultaneous application of collagen and the compounds enhancing the cell response of Th2 or Th3 lymphocytes such as TGF-beta, **IL-2**, **antibodies** to IL-12 which can augment the oral tolerance. In clinical praxis the treatment of osteoarthrosis with collagen type I has also been successfully applied. Induction of oral tolerance is new approach in the treatment of rheumatoid arthritis and as each new therapy, it requires refinement. In the future it is expected that an improved study design and a better understanding of the underlying mechanisms of oral tolerance will lead to an increased efficacy of the therapy in humans similar to the effectiveness previously demonstrated in animal models.

L3 ANSWER 2 OF 15 MEDLINE on STN
AN 1999262266 MEDLINE
DN 99262266 PubMed ID: 10330268
TI IL-2 may be a limiting factor precluding lymphocytes from genetically resistant mice from responding to HgCl2.
AU Jiang Y; Moller G
CS Department of Immunology, Wenner-Gren Institute, Arrhenius Laboratories for Natural Sciences, Stockholm University, 106 91 Stockholm, Sweden.
SO INTERNATIONAL IMMUNOLOGY, (1999 May) 11 (5) 627-33.
Journal code: 8916182. ISSN: 0953-8178.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199906
ED Entered STN: 19990712
Last Updated on STN: 19990712
Entered Medline: 19990623
AB It is unclear how HgCl2 causes autoimmune disorders in genetically predisposed rodents. We investigated the cytokine profile induced by HgCl2 in vitro, and found a high frequency of IL-2-secreting cells in splenocytes from susceptible A.SW and BALB/c mice, whereas the frequency was low in cells from resistant DBA/2 mice. More IL-2-secreting cells were induced in splenocytes from the high responder A.SW mice than in cells from the intermediate responder BALB/c mice. Unexpectedly, a similar level of IL-4 production was induced in splenocytes from BALB/c and DBA/2 mice. IL-4 production was high in unstimulated cells from A.SW mice and was further increased by HgCl2. IFN-gamma-secreting cells were detectable in splenocytes from all three strains after activation by HgCl2. The highest frequency of **IL-10**-secreting cells was found in splenocytes from A.SW mice after activation, whereas the frequency was lower in cells from BALB/c mice, followed by cells from DBA/2 mice. We showed that neutralizing anti-**IL-2** **antibody** profoundly inhibited the in vitro response to HgCl2. In contrast, **antibodies** against IL-4, IFN-gamma and **IL-10** did not significantly affect the response of splenocytes from either A.SW or DBA/2 mice. The addition of IL-2 into cultures enhanced the proliferative response to HgCl2 in splenocytes from DBA/2 mice to a level comparable with that in cells from BALB/c mice. We found no evidence for the suggestion that HgCl2 induces a Th1/Th2 imbalance in resistant/susceptible strains. We conclude that IL-2 may be a limiting factor precluding lymphocytes from resistant mice from responding to HgCl2.

L3 ANSWER 3 OF 15 MEDLINE on STN
AN 94001805 MEDLINE

DN 94001805 PubMed ID: 8104472
TI Brucella abortus induces a novel cytokine gene expression pattern characterized by elevated IL-10 and IFN-gamma in CD4+ T cells.
AU Svetic A; Jian Y C; Lu P; Finkelman F D; Gause W C
CS Department of Microbiology, Uniformed Services University of the Health Sciences, Bethesda, MD 20814.
NC AI21328 (NIAID)
SO INTERNATIONAL IMMUNOLOGY, (1993 Aug) 5 (8) 877-83.
Journal code: 8916182. ISSN: 0953-8178.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199310
ED Entered STN: 19940117
Last Updated on STN: 19950206
Entered Medline: 19931028
AB Immunization of BALB/c mice with killed Brucella abortus (BA) has previously been shown to increase serum IgG2a levels and long-term T cell clones from these mice secrete Th1-associated cytokines: IFN-gamma and IL-2 but not IL-4 or IL-5. We analyzed cytokine gene expression following primary immunization with BA to determine when CD4+ T cells first express cytokine genes and whether specific hypothesized cytokine patterns (e.g. Th precursor, Th0) could be identified prior to a Th1-like pattern. Our results demonstrated a highly consistent and novel pattern of Th1/Th2 cytokine gene expression characterized by elevated **IL-10** and IFN-gamma in CD4+ T cells which rapidly manifests itself and is sustained for at least 10 days after immunization. No elevation in IL-2 cytokine gene expression was observed and treatment of BA-immunized mice with blocking anti-**IL-2 antibodies** had no effect on the cytokine gene expression pattern, although treatment with anti-IFN **antibodies** resulted in increased IL-4, IL-5, and IL-9 cytokine gene expression, in the absence of any change in IFN-gamma or **IL-10** as early as 4 days after immunization. These results suggest that a whole pathogen may trigger sufficient costimulatory signals to rapidly induce effector T cells in the absence of elevated IL-2 and that **IL-10** is specifically elevated in certain Th1-like responses.

L3 ANSWER 4 OF 15 MEDLINE on STN
AN 93294307 MEDLINE
DN 93294307 PubMed ID: 8099937
TI Regulation of IL-5 in onchocerciasis. A critical role for IL-2.
AU Steel C; Nutman T B
CS Laboratory of Parasitic Diseases, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, MD 20892.
SO JOURNAL OF IMMUNOLOGY, (1993 Jun 15) 150 (12) 5511-8.
Journal code: 2985117R. ISSN: 0022-1767.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199307
ED Entered STN: 19930806
Last Updated on STN: 19950206
Entered Medline: 19930720
AB The cytokine profiles of PBMC obtained from individuals "immune" to Onchocerca volvulus infection were compared to those from infected individuals. The immune individuals had significantly higher levels of both IL-2 and IL-5 in response to parasite Ag than did those individuals with active infection (mean IL-2 = 1.3 and 0.138 U/ml, respectively; mean IL-5 = 973 and 147.4 pg/ml, respectively), and there was a direct correlation between the production of IL-2 and IL-5. To examine the mechanism underlying the possible association between these two cytokines

in patients infected with onchocerciasis, reverse transcription followed by polymerase chain reaction was used to measure IL-5 mRNA. In response to rIL-2, IL-5 mRNA appeared as early as early as 3 h after stimulation of patient PBMC, reaching a peak at 24 h; further, this response was inhibited with neutralizing **antibodies** to IL-2. IL-2 was unable to induce mRNA expression for IL-4, IFN-gamma, **IL-10**, or granulocyte-macrophage-CSF. To assess whether IL-2 was specifically responsible for the up-regulation of Ag-induced IL-5 production in patients with onchocerciasis, IL-5 mRNA expression was measured in PBMC stimulated with parasite Ag. Up-regulation of IL-5 mRNA was seen in all patients (peaking at 72 h) in response to Ag stimulation and was found to be independent of proliferation to Ag; in addition, this up-regulation was specifically inhibited by neutralizing anti-**IL-2 antibodies**. Further, the primary source of IL-5 mRNA was determined to be CD4+ T cells. These findings suggest that IL-2 production is required to induce IL-5 and further implicates IL-5 as a possible mediator of protection in onchocerciasis.

L3 ANSWER 5 OF 15 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1999:367654 CAPLUS
DN 131:143361
TI IL-2 may be a limiting factor precluding lymphocytes from genetically resistant mice from responding to HgCl2
AU Jiang, Yun; Moller, Goran
CS Department of Immunology, Wenner-Gren Institute, Arrhenius Laboratories for Natural Sciences, Stockholm University, Stockholm, 106 91, Swed.
SO International Immunology (1999), 11(5), 627-633
CODEN: INIMEN; ISSN: 0953-8178
PB Oxford University Press
DT Journal
LA English
AB It is unclear how HgCl2 causes autoimmune disorders in genetically predisposed rodents. We investigated the cytokine profile induced by HgCl2 in vitro, and found a high frequency of IL-2-secreting cells in splenocytes from susceptible A.SW and BALB/c mice, whereas the frequency was low in cells from resistant DBA/2 mice. More IL-2-secreting cells were induced in splenocytes from the high responder A.SW mice than in cells from the intermediate responder BALB/c mice. Unexpectedly, a similar level of IL-4 production was induced in splenocytes from BALB/c and DBA/2 mice. IL-4 production was high in unstimulated cells from A.SW mice and was further increased by HgCl2. IFN-γ-secreting cells were detectable in splenocytes from all three strains after activation by HgCl2. The highest frequency of **IL-10**-secreting cells was found in splenocytes from A.SW mice after activation, whereas the frequency was lower in cells from BALB/c mice, followed by cells from DBA/2 mice. We showed that neutralizing anti-**IL-2 antibody** profoundly inhibited the in vitro response to HgCl2. In contrast, **antibodies** against IL-4, IFN-γ and **IL-10** did not significantly affect the response of splenocytes from either A.SW or DBA/2 mice. The addition of IL-2 into cultures enhanced the proliferative response to HgCl2 in splenocytes from DBA/2 mice to a level comparable with that in cells from BALB/c mice. We found no evidence for the suggestion that HgCl2 induces a Th1/Th2 imbalance in resistant/susceptible strains. We conclude that IL-2 may be a limiting factor precluding lymphocytes from resistant mice from responding to HgCl2.
RE.CNT 45 THERE ARE 45 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 6 OF 15 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1993:647694 CAPLUS
DN 119:247694
TI Brucella abortus induces a novel cytokine gene expression pattern characterized by elevated IL-10 and IFN-γ in CD4+ T cells

AU Svetic, Antonela; Jian, Y. C.; Lu, P.; Finkelman, F. D.; Gause, W. C.
CS Dep. Microbiol. Med., Univ. Health Sci., Bethesda, MD, 20814, USA
SO International Immunology (1993), 5(8), 877-83
CODEN: INIMEN; ISSN: 0953-8178
DT Journal
LA English
AB Immunization of BALB/c mice with killed B. abortus (BA) has previously been shown to increase serum IgG2a levels, and long-term T cell clones from these mice secrete Th1-associated cytokines: IFN-γ and IL-2 but not IL-4 or IL-5. The authors analyzed cytokine gene expression following primary immunization with BA to determine when CD4+ T cells first express cytokine genes and whether specific hypothesized cytokine patterns (e.g. Th precursor, Th0) could be identified prior to a Th1-like pattern. The authors' results demonstrated a highly consistent and novel pattern of Th1/Th2 cytokine gene expression characterized by elevated **IL-10** and IFN-γ in CD4+ T cells which rapidly manifests itself and is sustained for at least 10 days after immunization. No elevation in IL-2 cytokine gene expression was observed and treatment of BA-immunized mice with blocking anti-**IL-2 antibodies** had no effect on the cytokine gene expression pattern, although treatment with anti-IFN **antibodies** resulted in increased IL-4, IL-5, and IL-9 cytokine gene expression, in the absence of any change in IFN-γ or **IL-10** as early as 4 days after immunization. Thus, a whole pathogen may trigger sufficient costimulatory signals to rapidly induce effector T cells in the absence of elevated IL-2 and **IL-10** is specifically elevated in certain Th1-like responses.

L3 ANSWER 7 OF 15 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1993:537210 CAPLUS
DN 119:137210
TI Regulation of IL-5 in onchocerciasis. A critical role for IL-2
AU Steel, Cathy; Nutman, Thomas B.
CS Lab. Parasit. Dis., Natl. Inst. Allergy Infect. Dis., Bethesda, MD, 20892, USA
SO Journal of Immunology (1993), 150(12), 5511-18
CODEN: JOIMA3; ISSN: 0022-1767
DT Journal
LA English
AB The cytokine profiles of PBMC obtained from individuals immune to Onchocerca volvulus infection were compared to those from infected individuals. The immune individuals had higher levels of both IL-2 and IL-5 in response to parasite antigens (Ag) than did those individuals with active infection (mean IL-2 = 1.3 and 0.138 U/mL, resp.; mean IL-5 = 973 and 147.4 pg/mL, resp.), and there was a direct correlation between the production of IL-2 and IL-5. To examine the mechanism underlying the possible association between these 2 cytokines in patients with onchocerciasis, reverse transcription followed by polymerase chain reaction was used to measure IL-5 mRNA. In response to rIL-2, IL-5 mRNA appeared as early as 3 h after stimulation of patient PBMC, reaching a peak at 24 h; further, this response was inhibited with neutralizing **antibodies** to IL-2. IL-2 could not induce mRNA expression for IL-4, IFN-γ, **IL-10**, or granulocyte-macrophage-CSF. To assess whether IL-2 was specifically responsible for the up-regulation of Ag-induced IL-5 production in patients with onchocerciasis, IL-5 mRNA expression was measured in PBMC stimulated with parasite Ag. Up-regulation of IL-5 mRNA was seen in all patients (peaking at 72 h) in response to Ag stimulation and was found to be independent of proliferation to Ag; in addition, this up-regulation was specifically inhibited by neutralizing anti-**IL-2 antibodies**. Further, the primary source of IL-5 mRNA was determined to be CD4+ T cells. These findings suggest that IL-2 production is required to induce IL-5 and further implicate IL-5 as a possible mediator of protection in onchocerciasis.

L3 ANSWER 8 OF 15 USPATFULL on STN

AN 2004:1816 USPATFULL
TI Prevention or treatment of cancer using integrin alphavbeta3 antagonists in combination with other agents
IN Woessner, Richard, Lafayette, CO, UNITED STATES
Kiener, Peter, Doylestwon, PA, UNITED STATES
Dormitzer, Melissa, Germantown, MD, UNITED STATES
Walsh, William, Sharpsburg, MD, UNITED STATES
Heinrichs, Jon, North Potomac, MD, UNITED STATES
PA MedImmune, Inc. (U.S. corporation)
PI US 2004001835 A1 20040101
AI US 2003-379189 A1 20030304 (10)
PRAI US 2002-361859P 20020304 (60)
US 2002-370398P 20020405 (60)
US 2003-444265P 20030130 (60)
DT Utility
FS APPLICATION
LREP PENNIE AND EDMONDS, 1155 AVENUE OF THE AMERICAS, NEW YORK, NY, 100362711
CLMN Number of Claims: 44
ECL Exemplary Claim: 1
DRWN 8 Drawing Page(s)
LN.CNT 6588
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to methods and compositions designed for the treatment, management or prevention of cancer. The methods of the invention comprise the administration of an effective amount of one or more antagonists of Integrin α.sub.Vβ.sub.3 alone or in combination with the administration of an effective amount of one or more other agents useful for cancer therapy. The invention also provides pharmaceutical compositions comprising one or more antagonists of Integrin α.sub.Vβ.sub.3 and/or one or more other agents useful for cancer therapy. In particular, the invention is directed to methods of treatment and prevention of cancer by the administration of a therapeutically or prophylactically effective amount of one or more antagonists of Integrin α.sub.Vβ.sub.3 alone or in combination with standard and experimental therapies for treatment or prevention of cancer. Also included are methods for screening for epitope-specific Integrin α.sub.Vβ.sub.3 antagonists which can be used according to the methods of the invention. In addition, methods for facilitating the use of Integrin α.sub.Vβ.sub.3 antagonists in the analysis of Integrin α.sub.Vβ.sub.3 expression in biopsies of animal model and clinical study samples are also contemplated.

L3 ANSWER 9 OF 15 USPATFULL on STN
AN 2003:237907 USPATFULL
TI Compositions and methods for the therapy and diagnosis of colon cancer
IN King, Gordon E., Shoreline, WA, UNITED STATES
Meagher, Madeleine Joy, Seattle, WA, UNITED STATES
Xu, Jiangchun, Bellevue, WA, UNITED STATES
Secrist, Heather, Seattle, WA, UNITED STATES
Jiang, Yuqiu, Kent, WA, UNITED STATES
PA Corixa Corporation, Seattle, WA, UNITED STATES, 98104 (U.S. corporation)
PI US 2003166064 A1 20030904
AI US 2002-99926 A1 20020314 (10)
RLI Continuation-in-part of Ser. No. US 2001-33528, filed on 26 Dec 2001, PENDING Continuation-in-part of Ser. No. US 2001-920300, filed on 31 Jul 2001, PENDING
PRAI US 2001-302051P 20010629 (60)
US 2001-279763P 20010328 (60)
US 2000-223283P 20000803 (60)
DT Utility
FS APPLICATION
LREP SEED INTELLECTUAL PROPERTY LAW GROUP PLLC, 701 FIFTH AVE, SUITE 6300, SEATTLE, WA, 98104-7092

CLMN Number of Claims: 17
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 8531
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Compositions and methods for the therapy and diagnosis of cancer, particularly colon cancer, are disclosed. Illustrative compositions comprise one or more colon tumor polypeptides, immunogenic portions thereof, polynucleotides that encode such polypeptides, antigen presenting cell that expresses such polypeptides, and T cells that are specific for cells expressing such polypeptides. The disclosed compositions are useful, for example, in the diagnosis, prevention and/or treatment of diseases, particularly colon cancer.

L3 ANSWER 10 OF 15 USPATFULL on STN
AN 2003:219754 USPATFULL
TI Tissues or organs for use in xenotransplantation
IN Liljedahl, Monika, La Jolla, CA, UNITED STATES
Marcantonio, Daniela, San Diego, CA, UNITED STATES
Aspland, Simon Eric, San Diego, CA, UNITED STATES
PI US 2003153044 A1 20030814
AI US 2002-303686 A1 20021121 (10)
RLI Continuation-in-part of Ser. No. US 2002-147286, filed on 14 May 2002, PENDING
PRAI US 2001-291394P 20010514 (60)
US 2001-312125P 20010813 (60)
US 2002-367090P 20020321 (60)
DT Utility
FS APPLICATION
LREP KNOBBE MARTENS OLSON & BEAR LLP, 2040 MAIN STREET, FOURTEENTH FLOOR, IRVINE, CA, 92614
CLMN Number of Claims: 73
ECL Exemplary Claim: 1
DRWN 14 Drawing Page(s)
LN.CNT 5751
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides cells, tissues or organs for use in cell therapy or xenotransplantation in which at least one gene comprising an antigenic determinant recognized by a recipient organism has been disrupted. The present invention also includes methods of administering such cells and transplanting such tissues or organs in which genes encoding antigenic determinants recognized by the recipient organism have been disrupted.

L3 ANSWER 11 OF 15 USPATFULL on STN
AN 2003:134089 USPATFULL
TI Tissues or organs for use in xenotransplantation
IN Liljedahl, Monika, La Jolla, CA, UNITED STATES
Marcantonio, Daniela, San Diego, CA, UNITED STATES
Aspland, Simon Eric, San Diego, CA, UNITED STATES
PI US 2003092174 A1 20030515
AI US 2002-147286 A1 20020514 (10)
PRAI US 2001-291394P 20010514 (60)
US 2001-312125P 20010813 (60)
US 2002-367090P 20020321 (60)
DT Utility
FS APPLICATION
LREP KNOBBE MARTENS OLSON & BEAR LLP, 2040 MAIN STREET, FOURTEENTH FLOOR, IRVINE, CA, 92614
CLMN Number of Claims: 55
ECL Exemplary Claim: 1
DRWN 14 Drawing Page(s)
LN.CNT 3786
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention provides cells, tissues or organs for use in cell therapy or xenotransplantation in which at least one gene comprising an antigenic determinant recognized by a recipient organism has been disrupted. The present invention also includes methods of administering such cells and transplanting such tissues or organs in which genes encoding antigenic determinants recognized by the recipient organism have been disrupted.

L3 ANSWER 12 OF 15 USPATFULL on STN
AN 2003:106233 USPATFULL
TI Compositions and methods for the therapy and diagnosis of pancreatic cancer
IN Benson, Darin R., Seattle, WA, UNITED STATES
Kalos, Michael D., Seattle, WA, UNITED STATES
Lodes, Michael J., Seattle, WA, UNITED STATES
Persing, David H., Redmond, WA, UNITED STATES
Hepler, William T., Seattle, WA, UNITED STATES
Jiang, Yuqiu, Kent, WA, UNITED STATES
PA Corixa Corporation, Seattle, WA, UNITED STATES, 98104 (U.S. corporation)
PI US 2003073144 A1 20030417
AI US 2002-60036 A1 20020130 (10)
PRAI US 2001-333626P 20011127 (60)
US 2001-305484P 20010712 (60)
US 2001-265305P 20010130 (60)
US 2001-267568P 20010209 (60)
US 2001-313999P 20010820 (60)
US 2001-291631P 20010516 (60)
US 2001-287112P 20010428 (60)
US 2001-278651P 20010321 (60)
US 2001-265682P 20010131 (60)
DT Utility
FS APPLICATION
LREP SEED INTELLECTUAL PROPERTY LAW GROUP PLLC, 701 FIFTH AVE, SUITE 6300, SEATTLE, WA, 98104-7092
CLMN Number of Claims: 17
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 14253
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Compositions and methods for the therapy and diagnosis of cancer, particularly pancreatic cancer, are disclosed. Illustrative compositions comprise one or more pancreatic tumor polypeptides, immunogenic portions thereof, polynucleotides that encode such polypeptides, antigen presenting cell that expresses such polypeptides, and T cells that are specific for cells expressing such polypeptides. The disclosed compositions are useful, for example, in the diagnosis, prevention and/or treatment of diseases, particularly pancreatic cancer.

L3 ANSWER 13 OF 15 USPATFULL on STN
AN 2002:272801 USPATFULL
TI Compositions and methods for the therapy and diagnosis of colon cancer
IN Stolk, John A., Bothell, WA, UNITED STATES
Xu, Jiangchun, Bellevue, WA, UNITED STATES
Chenault, Ruth A., Seattle, WA, UNITED STATES
Meagher, Madeleine Joy, Seattle, WA, UNITED STATES
PA Corixa Corporation, Seattle, WA, UNITED STATES, 98104 (U.S. corporation)
PI US 2002150922 A1 20021017
AI US 2001-998598 A1 20011116 (9)
PRAI US 2001-304037P 20010710 (60)
US 2001-279670P 20010328 (60)
US 2001-267011P 20010206 (60)
US 2000-252222P 20001120 (60)
DT Utility
FS APPLICATION

LREP SEED INTELLECTUAL PROPERTY LAW GROUP PLLC, 701 FIFTH AVE, SUITE 6300, SEATTLE, WA, 98104-7092
CLMN Number of Claims: 17
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 9233
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Compositions and methods for the therapy and diagnosis of cancer, particularly colon cancer, are disclosed. Illustrative compositions comprise one or more colon tumor polypeptides, immunogenic portions thereof, polynucleotides that encode such polypeptides, antigen presenting cell that expresses such polypeptides, and T cells that are specific for cells expressing such polypeptides. The disclosed compositions are useful, for example, in the diagnosis, prevention and/or treatment of diseases, particularly colon cancer.

L3 ANSWER 14 OF 15 USPATFULL on STN
AN 2002:243051 USPATFULL
TI Compositions and methods for the therapy and diagnosis of ovarian cancer
IN Algate, Paul A., Issaquah, WA, UNITED STATES
Jones, Robert, Seattle, WA, UNITED STATES
Harlocker, Susan L., Seattle, WA, UNITED STATES
PA Corixa Corporation, Seattle, WA, UNITED STATES, 98104 (U.S. corporation)
PI US 2002132237 A1 20020919
AI US 2001-867701 A1 20010529 (9)
PRAI US 2000-207484P 20000526 (60)
DT Utility
FS APPLICATION
LREP SEED INTELLECTUAL PROPERTY LAW GROUP PLLC, 701 FIFTH AVE, SUITE 6300, SEATTLE, WA, 98104-7092
CLMN Number of Claims: 11
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 25718
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Compositions and methods for the therapy and diagnosis of cancer, particularly ovarian cancer, are disclosed. Illustrative compositions comprise one or more ovarian tumor polypeptides, immunogenic portions thereof, polynucleotides that encode such polypeptides, antigen presenting cell that expresses such polypeptides, and T cells that are specific for cells expressing such polypeptides. The disclosed compositions are useful, for example, in the diagnosis, prevention and/or treatment of diseases, particularly ovarian cancer.

L3 ANSWER 15 OF 15 USPATFULL on STN
AN 2000:87729 USPATFULL
TI Method of converting a Th2-type allergic immune response into a Th1-type immune response
IN DeKruyff, Rosemarie H., Stanford, CA, United States
Umetsu, Dale T., Stanford, CA, United States
PA The Board of Trustees of the Leland Stanford Junior University, Palo Alto, CA, United States (U.S. corporation)
PI US 6086898 20000711
AI US 1999-339068 19990623 (9)
PRAI US 1998-90390P 19980623 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Chan, Christina Y.; Assistant Examiner: Ewoldt, Gerald R.
LREP Bozicevic, Field & Francis, Sherwood, Pamela
CLMN Number of Claims: 19
ECL Exemplary Claim: 1
DRWN 17 Drawing Figure(s); 10 Drawing Page(s)
LN.CNT 1767

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Methods are provided for the treatment of allergic and other immune disorders associated with overproduction of Th2 type cytokines by antigen specific T cells. Immunotherapy with adjuvants, as provided in the present invention, greatly inhibits the development of airway hyperreactivity and airway inflammation. Such immunotherapy is shown to reverse ongoing airway disease, and convert allergic inflammatory responses into protective immune responses. Conditions of particular interest include allergic conditions associated with production of Th2 cytokines and/or IgE antibodies, asthma, allergic rhinitis, and anaphylactic reactions. The addition of adjuvant induces a Th1-type immune response and can redirect an established Th2-type response to a Th1-type response for the selected antigen. Preferably, antigen-specific IgE production is reduced without altering the intensity of the antigen-specific proliferative response. One particularly preferred adjuvant for use in accordance with the present invention is a Listeria adjuvant.

=> d his

(FILE 'HOME' ENTERED AT 09:34:00 ON 17 FEB 2004)

FILE 'MEDLINE, CAPLUS, USPATFULL' ENTERED AT 09:34:13 ON 17 FEB 2004

L1 5599 S IL-10 (P) (ANTIBOD?)

L2 2256 S L1 (P) (IL-2)

L3 15 S L1 (P) (IL-2 ANTIBOD?)

=> d l3 1-15 kwic

L3 ANSWER 1 OF 15 MEDLINE on STN

AB . . . connected with induction of regulatory T cells of Th2/Th3 phenotypes in gut with characteristic profile of anti-inflammatory cytokines as IL-4, **IL-10** and TGF-beta. By means of circulation the lymphocytes enter the affected place and when meeting again with the antigen, they. . . the simultaneous application of collagen and the compounds enhancing the cell response of Th2 or Th3 lymphocytes such as TGF-beta, **IL-2**, **antibodies** to IL-12 which can augment the oral tolerance. In clinical praxis the treatment of osteoarthrosis with collagen type I has. . .

L3 ANSWER 2 OF 15 MEDLINE on STN

AB . . . by HgCl2. IFN-gamma-secreting cells were detectable in splenocytes from all three strains after activation by HgCl2. The highest frequency of **IL-10**-secreting cells was found in splenocytes from A.SW mice after activation, whereas the frequency was lower in cells from BALB/c mice, followed by cells from DBA/2 mice. We showed that neutralizing anti-**IL-2 antibody** profoundly inhibited the in vitro response to HgCl2. In contrast, **antibodies** against IL-4, IFN-gamma and **IL-10** did not significantly affect the response of splenocytes from either A.SW or DBA/2 mice. The addition of IL-2 into cultures. . .

L3 ANSWER 3 OF 15 MEDLINE on STN

AB . . . a Th1-like pattern. Our results demonstrated a highly consistent and novel pattern of Th1/Th2 cytokine gene expression characterized by elevated **IL-10** and IFN-gamma in CD4+ T cells which rapidly manifests itself and is sustained for at least 10 days after immunization. No elevation in IL-2 cytokine gene expression was observed and treatment of BA-immunized mice with blocking anti-**IL-2 antibodies** had no effect on the cytokine gene expression pattern, although treatment with anti-IFN **antibodies** resulted in increased IL-4, IL-5, and IL-9 cytokine gene expression, in the absence of any change in IFN-gamma or **IL-10** as

early as 4 days after immunization. These results suggest that a whole pathogen may trigger sufficient costimulatory signals to rapidly induce effector T cells in the absence of elevated IL-2 and that **IL-10** is specifically elevated in certain Th1-like responses.

L3 ANSWER 4 OF 15 MEDLINE on STN
AB . . . 3 h after stimulation of patient PBMC, reaching a peak at 24 h; further, this response was inhibited with neutralizing **antibodies** to IL-2. IL-2 was unable to induce mRNA expression for IL-4, IFN-gamma, **IL-10**, or granulocyte-macrophage-CSF. To assess whether IL-2 was specifically responsible for the up-regulation of Ag-induced IL-5 production in patients with onchocerciasis,. . . stimulation and was found to be independent of proliferation to Ag; in addition, this up-regulation was specifically inhibited by neutralizing anti-**IL-2 antibodies**. Further, the primary source of IL-5 mRNA was determined to be CD4+ T cells. These findings suggest that IL-2 production. . .

L3 ANSWER 5 OF 15 CAPLUS COPYRIGHT 2004 ACS on STN
AB . . . by HgCl2. IFN-γ-secreting cells were detectable in splenocytes from all three strains after activation by HgCl2. The highest frequency of **IL-10**-secreting cells was found in splenocytes from A.SW mice after activation, whereas the frequency was lower in cells from BALB/c mice, followed by cells from DBA/2 mice. We showed that neutralizing anti-**IL-2 antibody** profoundly inhibited the in vitro response to HgCl2. In contrast, **antibodies** against IL-4, IFN-γ and **IL-10** did not significantly affect the response of splenocytes from either A.SW or DBA/2 mice. The addition of IL-2 into cultures. . .

L3 ANSWER 6 OF 15 CAPLUS COPYRIGHT 2004 ACS on STN
AB . . . Th1-like pattern. The authors' results demonstrated a highly consistent and novel pattern of Th1/Th2 cytokine gene expression characterized by elevated **IL-10** and IFN-γ in CD4+ T cells which rapidly manifests itself and is sustained for at least 10 days after immunization. No elevation in IL-2 cytokine gene expression was observed and treatment of BA-immunized mice with blocking anti-**IL-2 antibodies** had no effect on the cytokine gene expression pattern, although treatment with anti-IFN **antibodies** resulted in increased IL-4, IL-5, and IL-9 cytokine gene expression, in the absence of any change in IFN-γ or **IL-10** as early as 4 days after immunization. Thus, a whole pathogen may trigger sufficient costimulatory signals to rapidly induce effector T cells in the absence of elevated IL-2 and **IL-10** is specifically elevated in certain Th1-like responses.

L3 ANSWER 7 OF 15 CAPLUS COPYRIGHT 2004 ACS on STN
AB . . . 3 h after stimulation of patient PBMC, reaching a peak at 24 h; further, this response was inhibited with neutralizing **antibodies** to IL-2. IL-2 could not induce mRNA expression for IL-4, IFN-γ, **IL-10**, or granulocyte-macrophage-CSF. To assess whether IL-2 was specifically responsible for the up-regulation of Ag-induced IL-5 production in patients with onchocerciasis,. . . stimulation and was found to be independent of proliferation to Ag; in addition, this up-regulation was specifically inhibited by neutralizing anti-**IL-2 antibodies**. Further, the primary source of IL-5 mRNA was determined to be CD4+ T cells. These findings suggest that IL-2 production. . .

L3 ANSWER 8 OF 15 USPATFULL on STN
DETD . . . propranolol, and puromycin homologs, and cytoxan. Examples of non-chemotherapeutic immunomodulatory agents include, but are not limited to, anti-T cell receptor **antibodies** (e.g., anti-CD4 **antibodies** (e.g., cM-T412 (Boeringer), IDEC-CE9.1® (IDEC and SKB), mAB 4162W94, Orthoclone and OKTcdr4a (Janssen-Cilag)), anti-CD3

antibodies (e.g., Nuvion (Product Design Labs), OKT3 (Johnson & Johnson), or Rituxan (IDEC)), anti-CD5 **antibodies** (e.g., an anti-CD5 ricin-linked immunoconjugate), anti-CD7 **antibodies** (e.g., CHH-380 (Novartis)), anti-CD8 **antibodies**, anti-CD40 ligand monoclonal **antibodies** (e.g., IDEC-131 (IDEC)), anti-CD52 **antibodies** (e.g., CAMPATH 1H (Ilex)), anti-CD2 **antibodies** (e.g., MEDI-507 (Medimmune, Inc., International Publication Nos. WO 02/098370 and WO 02/069904), anti-CD11a **antibodies** (e.g., Xanelim (Genentech)), and anti-B7 **antibodies** (e.g., IDEC-114) (IDEC)); anti-cytokine receptor **antibodies** (e.g., anti-IFN receptor **antibodies**, anti-IL-2 receptor **antibodies** (e.g., Zenapax (Protein Design Labs)), anti-IL-4 receptor **antibodies**, anti-IL-6 receptor **antibodies**, anti-**IL-10** receptor **antibodies**, and anti-IL-12 receptor **antibodies**), anti-cytokine **antibodies** (e.g., anti-IFN **antibodies**, anti-TNF-α **antibodies**, anti-IL-1β **antibodies**, anti-IL-6 **antibodies**, anti-IL-8 **antibodies** (e.g., ABX-IL-8 (Abgenix)), anti-IL-12 **antibodies** and anti-IL-23 **antibodies**)); CTLA4-immunoglobulin; LFA-3TIP (Biogen, International Publication No. WO 93/08656 and U.S. Pat. No. 6,162,432); soluble cytokine receptors (e.g., the extracellular domain. . . IL-6 receptor or a fragment thereof); cytokines or fragments thereof (e.g., interleukin (IL)-2, IL-3, IL-4, IL-5, IL-6, IL-7, IL-8, IL-9, **IL-10**, IL-11, IL-12, IL-15, IL-23, TNF-α, TNF-β, interferon (IFN)-α, IFN-β, IFN-γ, and GM-CSF); and anti-cytokine **antibodies** (e.g., anti-**IL-2** **antibodies**, anti-IL-4 **antibodies**, anti-IL-6 **antibodies**, anti-**IL-10** **antibodies**, anti-IL-12 **antibodies**, anti-IL-15 **antibodies**, anti-TNF-α **antibodies** and anti-IFN-γ **antibodies**), and **antibodies** that immunospecifically bind to tumor-associated antigens (e.g., Herceptin®). In certain embodiments, an immunomodulatory agent is an immunomodulatory agent other than. . .

L3 ANSWER 9 OF 15 USPATFULL on STN

SUMM [2042] **For example**, certain amino **acids** may **be substituted for other** amino **acids in a protein** structure without appreciable loss **of interactive binding capacity** with structures **such as**, **for example**, antigen-binding regions of **antibodies or binding** sites on **substrate** molecules. Since **it is** the **interactive capacity and nature** of **a** protein that **defines that protein** 's biological functional activity, **certain amino acid sequence substitutions** can **be** made in **a protein sequence**, **and**, **of course**, **its underlying** DNA **coding** sequence, **and** nevertheless obtain **a** protein with like **properties**. It is thus **contemplated** that various changes may be made in the **peptide** sequences of the disclosed compositions, **or corresponding** **DNA** sequences which encode said peptides without appreciable loss **of** their biological **utility** or activity.

TABLE 1

Amino **Acids**	Codons

Alanine	Ala	A	GCA GCC GCG GCU
Cysteine	Cys	C	UGC UGU
Aspartic acid	Asp	**D**	**GAC GAU**
Glutamic acid	Glu	E	GAA **GAG**
Phenylalanine	Phe	**F**	UUC UUU
Glycine	Gly	G	GGA GGC GGG GGU
Histidine	His	H	CAC CAU
Isoleucine	Ile	I	AUA AUC AUU
Lysine	Lys	K	AAA AAG
Leucine	Leu	L	UUA UUG CUA CUC CUG CUU
Methionine. . . CCU			
Glutamine	Gln	Q	CAA CAG
Arginine	Arg	R	AGA AGG CGA CGC CGG CGU
Serine	Ser	S	AGC AGU **UCA** UCC UCG **UCU**
Threonine	Thr	T	ACA ACC ACG ACU
Valine	Val	V	GUA GUC GUG **GUU**
Tryptophan	Trp	W	UGG
Tyrosine	Tyr	Y	UAC UAU

L3 ANSWER 10 OF 15 USPATFULL on STN

DETD . . . Erythropoetin (EPO), anemic conditions; insulin: islets or the pancreas can be transplanted into diabetic patients; tumor necrosis factor α (TNF-alpha) **antibodies**, for example, for inflammatory diseases such as rheumatoid arthritis and Crohn's disease; **antibodies** against protein products encoded by oncogenes such as C-erbB-2, for example used for breast cancer and other cancers; anti-CD4 **antibodies** for example, for rheumatoid arthritis or psoriasis; anti-human Epidermal Growth Factor Receptor type 2 **antibodies**, for example, for breast cancer and other cancers; anti-Interleukin **antibodies**, such as anti-IL-1, anti-IL-8, anti **IL-10**, anti-IL-12 and anti-IL-15 to be used, for example, in inflammatory diseases, such as autoimmune diseases, rheumatoid arthritis, psoriasis, inflammatory bowel disease and in cancerous disease; anti-Interleukin 15 receptor anti-bodies for use against lymphoma and other malignancies, for example; anti-CD20 **antibodies** to be used, for example, for hemolytic anemia in autoimmune diseases and other hematopoetic disorders such as leukemia and lymphomas; anti-isotypic IGE **antibodies** for allergy; anti-LG914 **antibodies** for arteriosclerosis; Interferon-α for chronic hepatitis C, hairy cell leukemia and AIDS-related Kaposi's sarcoma and chronic myclogenous leukemia (CML), for. . . in donor animal such as pig of proteins, lipids and carbohydrates to induce tolerance in xenotransplantation; anti-CD40, CD28, CD25 and **IL-2 antibodies** and OKT3; anti-idiotypic **antibodies** against naturally formed **antibodies**; anti-isotypic IgG, IgM and IgA **antibodies**. The preceding is a

non exclusive list of some exemplary gene therapy applications.

L3 ANSWER 11 OF 15 USPATFULL on STN
DETD . . . Erythropoetin (EPO), anemic conditions; insulin: islets or the pancreas can be transplanted into diabetic patients; tumor necrosis factor α (TNF-alpha) **antibodies**, for example, for inflammatory diseases such as rheumatoid arthritis and Crohn's disease; **antibodies** against protein products encoded by oncogenes such as C-erbB-2, for example used for breast cancer and other cancers; anti-CD4 **antibodies** for example, for rheumatoid arthritis or psoriasis; anti-human Epidermal Growth Factor Receptor type 2 **antibodies**, for example, for breast cancer and other cancers; anti-Interleukin **antibodies**, such as anti-IL-1, anti-IL-8, anti **IL-10**, anti-IL-12 and anti-IL-15 to be used, for example, in inflammatory diseases, such as autoimmune diseases, rheumatoid arthritis, psoriasis, inflammatory bowel disease and in cancerous disease; anti-Interleukin 15 receptor anti-bodies for use against lymphoma and other malignancies, for example; anti-CD20 **antibodies** to be used, for example, for hemolytic anemia in autoimmune diseases and other hematopoetic disorders such as leukemia and lymphomas; anti-isotypic IGE **antibodies** for allergy; anti-LG914 **antibodies** for arteriosclerosis; Interferon-α for chronic hepatitis C, hairy cell leukemia and AIDS-related Kaposi's sarcoma and chronic myelogenous leukemia (CML), for. . . in donor animal such as pig of proteins, lipids and carbohydrates to induce tolerance in xenotransplantation; anti-CD40, CD28, CD25 and **IL-2 antibodies** and OKT3; anti-idiotypic **antibodies** against naturally formed **antibodies**; anti-isotypic IgG, IgM and IgA **antibodies**. The preceding is a non exclusive list of some exemplary gene therapy applications.

L3 ANSWER 12 OF 15 USPATFULL on STN
SUMM [2043] **SEQ ID** NO:2003 **is** the **determined cDNA sequence of** clone **61496359**

L3 ANSWER 13 OF 15 USPATFULL on STN
SUMM [2044] **SEQ ID** NO:1997 **is** the determined **cDNA sequence for** clone **62227174 R0394:B12**

L3 ANSWER 14 OF 15 USPATFULL on STN
SUMM [2043] **SEQ ID** NO: 2004 **represents** the **cDNA sequence for clone** AA165409.

L3 ANSWER 15 OF 15 USPATFULL on STN
DETD Anti-IFN-γ mAb R46A2 (HB170, ATCC), and anti-IL-4 mAb (11B11), were prepared from serum-free culture supernatants by ammonium sulfate precipitation. Monoclonal anti-**IL-2 antibody** S4B6 and anti-IFN-γ **antibody** XMG1.2 were obtained from Dr. Tim Mosmann (Univ. of Alberta, Edmonton, Canada). Anti-IL-4 mAb BVD4-1D11 and BVD6-24G2 were obtained from DNAX Research Institute, Palo Alto, Calif. Each of these **antibodies** was purified from ascites by ammonium sulfate precipitation and ion-exchange chromatography. Anti-**IL-10** mAb SXC.1 (DNAX) was purified by ammonium sulfate precipitation followed by Sepharose 4B chromatography. Anti-**IL-10** mAb 2A5 was purchased from Pharmingen (San Diego, Calif.). Neutralizing anti-IL-12 mAb C17.8 was purified from ascites by affinity chromatography.. . .